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| EXAMINER |
|---|
| HUYNH, PHUONG N |

| ART UNIT | PAPER NUMBER |
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| 1644 | 23 |

DATE MAILED: 07/30/2002

Please find below and/or attached an Office communication concerning this application or proceeding.

| Office Action Summary | Application No. | Applicant(s) | |
|---|---|---|---|
| | 09/292,053 | MICHELLE E. REFF | |
| | Examiner | Art Unit | |
| | " Neon" Phuong Huynh | 1644 | |

*-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --*

**Period for Reply**

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *Three* MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).
- Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

**Status**

1)☒ Responsive to communication(s) filed on *24 May 2002* .

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

**Disposition of Claims**

4)☒ Claim(s) *38-47* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *38-47* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

**Application Papers**

9)☐ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *04/19/99* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

11)☐ The proposed drawing correction filed on _____ is: a)☐ approved b)☐ disapproved by the Examiner.

If approved, corrected drawings are required in reply to this Office action.

12)☐ The oath or declaration is objected to by the Examiner.

**Priority under 35 U.S.C. §§ 119 and 120**

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____ .

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

14)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. § 119(e) (to a provisional application).

a) ☐ The translation of the foreign language provisional application has been received.

15)☐ Acknowledgment is made of a claim for domestic priority under 35 U.S.C. §§ 120 and/or 121.

**Attachment(s)**

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449) Paper No(s) _____
4) ☐ Interview Summary (PTO-413) Paper No(s). _____
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other

**DETAILED ACTION**

1. The request for continued examination under Rule 114 filed 5/24/02 is acknowledged.

2. Claims 38-47 are pending and are being acted upon in this Office Action.

3. The disclosure is objected to because of the following informality: (1) the specification disclosed on page 47, paragraph 1 and page 50 that SEQ ID NO: 1, 3, 5 and 7 are **primers** for PCR of the heavy and light chain variable domains of 6G5 and 5E8 while the paper copy of the sequence listing and computer readable form filed Sept 21, 2000 indicates that SEQ ID NO: 1, 3, 5 and 7 are polynucleotides encoding human polypeptides of SEQ ID NO: 2, 4, 6 and 8, respectively. (2) SEQ ID NO: is required on pages 50-52, 54-56, 59-60 and 62-63. Appropriate correction is required.

4. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

5. Claims 38-46 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling only for (1) a method of inhibiting IgE in a subject in need of such inhibition comprising administering an IgE inhibiting effective amount of an anti-human CD23 antibody comprising a human gamma-constant region wherein the anti-human CD23 antibody comprises a variable heavy domain having a sequence selected from the group consisting of the polypeptide of SEQ ID NO: 4 and SEQ ID NO: 8 encoded by a nucleic acid sequence of SEQ ID NO: 3 and 7; and a variable light domain having a sequence selected from the group consisting of the polypeptide of SEQ ID NO: 2 and SEQ ID NO: 6 encoded by a nucleic acid sequence selected from the group consisting of SEQ ID NO: 1 and 5 for treating an IgE mediated allergic disorder, (2) a method of inhibiting IgE in a subject in need of such inhibition comprising administering an IgE inhibiting effective amount of an anti-human CD23 antibody comprising a human gamma-constant region wherein the anti-human CD23 monoclonal antibody comprises a primate antigen-binding region or a rodent antigen-binding region for treating an IgE mediated allergic disorder, **does not** reasonably provide enablement for (1) any method mentioned above for treating any

disease. (2) a method of inhibiting IgE in a subject in need of such inhibition comprising administering an IgE inhibiting effective amount of an anti-human CD23 antibody comprising a human gamma-constant region wherein the anti-human CD23 monoclonal antibody comprises a primate antigen-binding "portion" or a rodent antigen-binding "portion". (3) a method of treating an IgE mediated allergic disorder in a human subject comprising administering an effective amount of any anti-human CD23 antibody comprising a human gamma-1 constant region for treating any disorder.

Factors to be considered in determining whether undue experimentation is required to practice the claimed invention are summarized *In re Wands* (858 F2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988)). The factors most relevant to this rejection are the scope of the claim, the amount of direction or guidance provided, the lack of sufficient working examples, the unpredictability in the art and the amount of experimentation required to enable one of skill in the art to practice the claimed invention. The specification disclosure is insufficient to enable one skilled in the art to practice the invention as broadly claimed without an undue amount of experimentation.

The specification discloses only a method of making various chimeric primate anti-human CD23 antibodies such as 5E8 and 6G5 containing a human gamma 1 constant region and a method of inhibiting IgE by administering an anti-human CD23 antibody comprising a human gamma-constant region wherein the anti-human CD23 antibody comprises a variable heavy domain having a sequence selected from the group consisting of the polypeptide of SEQ ID NO: 4 and SEQ ID NO: 8 encoded by a nucleic acid sequence of SEQ ID NO: 3 and 7; and a variable light domain having a sequence selected from the group consisting of the polypeptide of SEQ ID NO: 2 and SEQ ID NO: 6 encoded by a nucleic acid sequence selected from the group consisting of SEQ ID NO: 1 and 5 for treating an IgE mediated allergic disorder. The specification discloses that the claimed anti-CD23 antibodies can be used to treat any disease such as the ones listed on page 81 and continuing through page 84. The diseases such as transplant rejection, autoimmune and inflammatory diseases are allegedly can be treated by the claimed anti-CD23 antibodies. However, there is no disclosure in the specification as filed for treating or preventing any disease with autoimmune or inflammatory response.

The specification does not teach how to use *any* of the claimed anti-human CD23 antibodies for a method of inhibiting IgE in a human subject for the prevention of *any* autoimmune, inflammatory disease, including transplant rejection. There is insufficient guidance

as to the primate antigen binding "portion" or the rodent antigen-binding "portion" since the term "portion" does not convey the specific structure, such as the specific amino acid residues. The term "portion" could be as little as a single amino acid or it could be as long as 100 amino acids in length. Given the indefinite number of undisclosed amino acid sequence in relation to the "portion", it is unpredictable which undisclosed portion would bind human CD23, in turn, would be useful for any purpose.

Other than the specific chimeric primate anti-human CD23 antibodies mentioned above for a method of treating an IgE mediated allergic disorder, there are insufficient in vivo working examples in the specification that the claimed anti-human CD23 antibodies are effective in preventing or treating any autoimmune, chronic inflammatory diseases such as the ones listed on page 81 through page 84, including transplant rejection.

The Merck Manual (of record) teaches that ankylosing spondylitis, which is one of the diseases disclosed on page 81 of the instant specification as being a disease preventable or treatable with the anti-CD23 antibodies, can be treated by NSAIDS, cortisosteroid and radiotherapy to the spine. The Merck Manual (of record) does not teach any method of prevention for ankylosing spondylitis (See Merck Manual page 1334-1337). The Merck Manual does not teach a preventive method for rheumatoid arthritis which is one of the diseases disclosed on page 83 of instant specification as being preventable or treatable with the claimed anti-CD23 antibodies (See Merck Manual page 1305-1310).

For these reasons, it would require undue experimentation of one skilled in the art to practice the claimed invention. See page 1338, footnote 7 of Ex parte Aggarwal, 23 USPQ2d 1334 (PTO Bd. Pat App. & Inter. 1992). In re wands, 858 F.2d at 737, 8 USPQ2d at 1404 (Fed. Cir. 1988), the decision of the court indicates that the more unpredictable the area is, the more specific enablement is necessary. In view of the quantity of experimentation necessary, the limited working examples, the unpredictability of the art, the lack of sufficient guidance in the specification and the breadth of the claims, it would take an undue amount of experimentation for one skilled in the art to practice the claimed invention.

Applicant's arguments in the amendment filed 12/4/01 have been fully considered but are not persuasive.

Applicants' position is that (1) claims 38-46 are directed to a method for inhibiting IgE in vivo by administration of an anti-human CD23 antibody having human gamma-1 constant region, (2) the examiner has conceded on record that antibodies according to the invention possess IgE

inhibiting activity in vivo and there is no basis for maintaining the 112 enablement rejection against claims 38-46.

However, there are no in vivo working examples in the specification that the claimed anti-human CD23 antibodies are effective in preventing or treating any autoimmune, chronic inflammatory diseases and transplant rejection mediated by IgE in a human subject. Further, there is insufficient guidance as to the "portion" of primate antigen binding domain or the "portion" of a rodent antigen-binding domain that could bind human CD23 since the term "portion" does not convey the specific structure, and the specific amino acid residues. The term "portion" could be as little as a single amino acid or it could be as long as 100 amino acids. Given the indefinite number of undisclosed amino acid sequence in relation to the "portion", it is unpredictable which undisclosed portion would bind human CD23, in turn, would be useful for any purpose.

6. Claims 38-46 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor, at the time the application was filed, had possession of the claimed invention.

The specification does not reasonably provide a **written description** of a method of inhibiting IgE in a subject in need of such inhibition comprising administering an IgE inhibiting effective amount of an anti-human CD23 antibody comprising a human gamma-constant region wherein the anti-human CD23 antibody comprises a variable heavy domain having a sequence selected from the group consisting of the polypeptide of SEQ ID NO: 4 and SEQ ID NO: 8 encoded by a nucleic acid sequence of SEQ ID NO: 3 and 7; and a variable light domain having a sequence selected from the group consisting of the polypeptide of SEQ ID NO: 2 and SEQ ID NO: 6 encoded by a nucleic acid sequence selected from the group consisting of SEQ ID NO: 1 and 5 for treating any disorder such as any autoimmune, chronic inflammatory diseases and transplant rejection. (2) a method of inhibiting IgE in a subject in need of such inhibition comprising administering an IgE inhibiting effective amount of an anti-human CD23 antibody comprising a human gamma-constant region wherein the anti-human CD23 monoclonal antibody comprises a primate antigen-binding "portion" or a rodent antigen-binding "portion" for preventing or treating any disorder such as any autoimmune, chronic inflammatory diseases and transplant rejection.

The specification discloses only a method of making various chimeric primate anti-human CD23 antibodies such as 5E8 and 6G5 containing a human gamma 1 constant region and a method of inhibiting IgE by administering an anti-human CD23 antibody comprising a human gamma-constant region wherein the anti-human CD23 antibody comprises a variable heavy domain having a sequence selected from the group consisting of the polypeptide of SEQ ID NO: 4 and SEQ ID NO: 8 encoded by a nucleic acid sequence of SEQ ID NO: 3 and 7; and a variable light domain having a sequence selected from the group consisting of the polypeptide of SEQ ID NO: 2 and SEQ ID NO: 6 encoded by a nucleic acid sequence selected from the group consisting of SEQ ID NO: 1 and 5 for treating an IgE mediated allergic disorder. The specification discloses that the claimed anti-CD23 antibodies can be used to treat any disease such as the ones listed on page 81 and continuing through page 84. The diseases such as transplant rejection, autoimmune and inflammatory diseases are allegedly can be treated by the claimed anti-CD23 antibodies. However, there is no disclosure in the specification as filed for treating or preventing any autoimmune or inflammatory disease.

With the exception of the specific anti-CD23 antibodies for a method of treating an IgE mediated allergic disorder, there is insufficient written description about the structure associated with function of *any* primate antigen binding "portion", *any* rodent antigen-binding "portion", and *any* anti-human CD23 antibody comprising a human gamma-1 constant region for preventing or treating *any* disease. The term "portion" does not convey the specific structure such as the specific amino acid residues associated with function. The term "portion" could be as little as a single amino acid or it could be as long as 100 amino acids.

With regard to a method of inhibiting IgE in a subject in need of such inhibition comprising administering an IgE inhibiting effective amount of an anti-human CD23 antibody comprising a human gamma-constant region wherein the anti-human CD23 antibody comprises a variable heavy domain having a sequence selected from the group consisting of the polypeptide of SEQ ID NO: 4 and SEQ ID NO: 8 encoded by a nucleic acid sequence of SEQ ID NO: 3 and 7; and a variable light domain having a sequence selected from the group consisting of the polypeptide of SEQ ID NO: 2 and SEQ ID NO: 6 encoded by a nucleic acid sequence selected from the group consisting of SEQ ID NO: 1 and 5, the specification discloses on page 47, paragraph 1 and page 50 that SEQ ID NO: 1, 3, 5 and 7 are **primers** for PCR of the heavy and light chain variable domains of 6G5 and 5E8 and not the variable heavy and light domains, which is conflicting with the sequence listing in paper copy and computable readable form. Further, the

specification discloses a method of inhibiting only IgE mediated allergic disorder using the specific anti-CD23 antibody, the method of inhibiting any IgE mediated disorders comprising administering any anti-human CD23 antibody comprising a human gamma-1 constant region is not adequately described. One of skill in the art would therefore conclude that the specification fails to disclose a representative number of species to describe the claimed genus. See *Eli Lilly*, 119F.3d 1559, 43 USPQ2d 1398. Thus, Applicant was not in possession of the claimed genus. *See University of California v. Eli Lilly and Co. 43 USPQ2d 1398.*

Applicant is directed to the Revised Interim Guidelines for the Examination of Patent Applications Under the 35 U.S.C. 112, ¶ 1 "Written Description" Requirement, Federal Register, Vol. 66, No. 4, pages 1099-1111, Friday January 5, 2001.

7. The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter, which the applicant regards as his invention.

8. Claims 39 and 41 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

The recitation of "portion" in claims 39 and 41 is indefinite and ambiguous because a single amino acid would still be considered a portion. One of ordinary skill in the art cannot appraise the metes and bounds of the claimed limitation.

9. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office Action:

A person shall be entitled to a patent unless –

(a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

10. Claims 38, 40-45 and 47 are rejected under 35 U.S.C. 102(a) as being anticipated by the WO 96/12741 publication (of record, May 1996, PTO 1449) as evidence by Saxon et al (of record, J Immunol 147(11): 4000-4006, 1991; PTO 1449).

The WO 96/12741 publication teaches a method of inhibiting allergy in a patient comprising administering a pharmaceutical effective amount of monoclonal humanized anti-CD 23 antibody that contains a rodent binding portion and a human gamma 1 constant region (See claims 20 and 11-15, page 4, lines 1-3, and lines 15-19, lines in particular). The reference anti-

CD23 antibody inhibits IgE production and IgE mediated B cell differentiation in vivo (See page 8, line 25-28, claims 1-4 in particular). Further, the WO 96/12741 publication teaches a method of inhibiting allergy in a patient using chimeric and humanized anti-CD23 antibody and the reference humanized antibody contains IgG 1 constant region (See claims 20 and 11-15, page 4, lines 1-3, and lines 15-19, lines in particular). It is an inherent property of anti-CD23 antibodies to inhibit IgE expression induced by IL-4 as evidence by Saxon et al (See abstract, and page 4004, column 1, in particular). Claim 43 is included in this rejection because the ability of the reference anti-CD23 antibody to inhibit IgE expression in vivo is an inherent property and the anti-CD23 would also inhibit the IgE expression in vitro. Thus, the reference teachings anticipate the claimed invention.

Applicant's arguments in the amendment filed 12/4/01 have been fully considered but are not persuasive.

Applicants' position is that (1) the WO 96/12741 does not anticipate the claimed method given the myriad of different possible CD23 agonist and antagonist including antibodies, antibody fragments, single chains, chimeric and humanized antibodies for a variety of potential application including treatment of allergic disorders, autoimmune disorders and inflammatory disorders, (2) the reference does not provide the requisite specific incentive to specifically selected as the CD23 antagonist or agonist, and (3) the Saxon et al reference discloses that anti-CD23 antibodies inhibit IgE expression but does not substantiate a conclusion that one of ordinary skill would have been specifically motivated to select an anti-human CD23 antibody containing human gamma 1 constant domains for therapy.

However, even without the Saxon et al reference, the WO 96/12741 publication still teaches that the reference anti-CD23 antibody inhibits IgE production and IgE mediated B cell differentiation in vivo (See page 8, line 25-28, claims 1-4 in particular).

11. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 103(a) that form the basis for the rejections under this section made in this Office Action:

A person shall be entitled to a patent unless:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

12. This application currently names joint inventors. In considering Patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

13. Claims 38-39 and 43 are rejected under 35 U.S.C. 103(a) as being unpatentable over the WO 96/12741 publication (of record, May 1996, PTO 1449) in view of Saxon et al (of record, J Immunol 147(11): 400-4006, 1991; PTO 1449) and US Pat No. 5,658,570 (of record, Aug 1997, PTO 892).

The teachings of the WO 96/12741 publication have been discussed supra.

The claimed invention as recited in claim 39 differs from the reference only that the anti-human CD 23 monoclonal antibody comprises a primate antigen-binding portion.

The claimed invention as recited in claim 43 differs from the reference only that the anti-human CD23 monoclonal antibody inhibits IgE expression in vitro.

The '570 patent teaches chimeric or humanized anti-CD23 antibodies which comprise a human constant region of IgG isotype and a primate antigen binding region (See claims 1-8, and column 8, lines 52-53) and a method of administering a therapeutically amount of said antibodies (See column 6, lines 1-8, in particular). The '570 patent teaches the antibody constant region derived from a human to ensure that they appear more human-like so that the probability of adverse reaction is lessened (See column 5, lines 32-38, in particular).

Saxon *et al* teach anti-CD23 antibodies inhibit IgE expression (See abstract, page 4002, column 2, Suppression of ongoing IgE production by FcεII (CD23) mab, in particular).

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to substitute the anti-CD23 antibody as taught by the WO 96/12741 publication for the anti-human CD23 monoclonal antibody comprises a primate antigen binding portion as taught by The '570 patent for a method of inhibiting IgE in a human subject as taught by the WO 96/12741 publication and Saxon *et al*. From the combined teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention.

One having ordinary skill in the art would have been motivated to do this because the '570 patent teaches the antibody constant region derived from a human would ensure that the antibody appears more human-like so that the probability of adverse reaction is lessened (See column 5, lines 32-38, in particular). The WO 96/12741 publication teaches chimeric humanized anti-CD 23 antibody is useful for inhibiting IgE production and IgE mediated B cell differentiation in vivo for a method of treating IgE mediated allergic disorders (See claims 20 and 11-15, page 4, lines 1-3, and lines 15-19, lines in particular). Saxon *et al* teach anti-CD23 antibodies inhibit IgE expression (See abstract, page 4002, column 2, Suppression of ongoing IgE production by FcεII (CD23) mab, in particular).

Applicant's arguments in the amendment filed 12/4/01 have been fully considered but are not persuasive.

Applicants' position is that (1) the WO 96/12741 does not anticipates the claimed method given the myriad of different possible CD23 agonist and antagonist including antibodies, antibody fragments, single chains, chimeric and humanized antibodies for a variety of potential application including treatment of allergic disorders, autoimmune disorders and inflammatory disorders, (2) the reference does not provide the requisite specific incentive to specifically selected as the CD23 antagonist or agonist, (3) the Saxon et al reference discloses that anti-CD23 antibodies inhibit IgE expression but does not substantiate a conclusion that one of ordinary skill would have been specifically motivated to select an anti-human CD23 antibody containing human gamma 1 constant domains for therapy, (4) the addition of Newman patent does not cure the deficiencies of the rejection since the patent fails to suggest the intrinsic advantages of anti-CD23 antibodies having gamma 1 effector function for therapy.

However, even without the Saxon et al reference, the WO 96/12741 publication still teaches that the reference anti-CD23 antibody inhibits IgE production and IgE mediated B cell differentiation in vivo (See page 8, line 25-28, claims 1-4 in particular). Further, the WO 96/12741 publication teaches a method of inhibiting allergy in a patient using chimeric and humanized anti-CD23 antibody and the reference humanized antibody contains IgG 1 constant region (See claims 20 and 11-15, page 4, lines 1-3, and lines 15-19, lines in particular).

In response to applicant's argument that the references fail to show certain features of applicant's invention, it is noted that the features upon which applicant relies (i.e., the intrinsic advantages of anti-CD23 antibodies having gamma effector function for therapy) are not recited in the rejected claim(s). Although the claims are interpreted in light of the specification,

limitations from the specification are not read into the claims. See *In re Van Geuns*, 988 F.2d 1181, 26 USPQ2d 1057 (Fed. Cir. 1993).

14. Claim 46 appears to be free of art.

15. No claim is allowed.

16. Any inquiry concerning this communication or earlier communications from the examiner should be directed to "Neon" Phuong Huynh whose telephone number is (703) 308-4844. The examiner can normally be reached Monday through Friday from 9:00 am to 6:00 p.m. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Christina Chan can be reached on (703) 308-3973. Any inquiry of a general nature or relating to the status of this application should be directed to the Technology Center 1600 receptionist whose telephone number is (703) 308-0196.

17. Papers related to this application may be submitted to Technology Center 1600 by facsimile transmission. Papers should be faxed to Technology Center 1600 via the PTO Fax Center located in Crystal Mall 1. The faxing of such papers must conform to the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). The CM1 Fax Center telephone number is (703) 305-7401.

Phuong N. Huynh, Ph.D.
Patent Examiner
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July 29, 2002

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